What is claimed:

1. A method of determining whether a compound inhibits HIV-1 reverse transcriptase which comprises:

a) contacting a yeast cell with the compound, which cell comprises (i) a first plasmid which expresses a fusion protein comprising a p66 subunit polypeptide of HIV-1 reverse transcriptase, (ii) a second plasmid which expresses a fusion protein comprising a p51 subunit polypeptide of HIV-1 reverse transcriptase, and (iii) a reporter gene which is activated in the presence of a complex between the p66 subunit polypeptide and the p51 subunit polypeptide, and determining the level of activity of the reporter gene in the cell in the presence of the compound; and

b) comparing the level of activity of the reporter gene determined in step (a) with a level of activity of the reporter gene determined in the absence of the compound, wherein a decreased level of activity of the reporter gene in step (a) indicates that the compound inhibits formation of a complex between the p51 subunit polypeptide of HIV-1 reverse transcriptase and the p66 subunit polypeptide of HIV-1 reverse transcriptase, thereby indicating that the compound inhibits HIV-1 reverse transcriptase.

2. A method of determining whether a compound inhibits formation of a complex between a p66 subunit polypeptide of HIV-1 reverse transcriptase and a p51 subunit polypeptide of HIV-1 reverse transcriptase which comprises:

a) contacting a yeast cell with the compound, which cell comprises (i) a first plasmid which expresses a fusion protein comprising a p66 subunit polypeptide of HIV-1 reverse transcriptase, (ii) a second plasmid which expresses a fusion protein comprising a p51 subunit polypeptide of HIV-1 reverse transcriptase, and (iii) a reporter gene which is activated in the presence of a complex between the p66 subunit polypeptide and the p51 subunit polypeptide, and determining the level of activity of the reporter gene in the cell in the presence of the compound; and

b) comparing the level of activity of the reporter gene determined in step (a) with a level of activity of the reporter gene determined in the absence of the compound, wherein a decreased level of activity of the reporter gene in step (a) indicates that the compound inhibits formation of a complex between the p51 subunit polypeptide of HIV-1 reverse transcriptase and the p66 subunit polypeptide of HIV-1 reverse transcriptase.

3. A method of determining whether a compound inhibits HIV-1 reverse transcriptase which comprises:

a) contacting a yeast cell with the compound, which cell comprises (i) a first plasmid which expresses a fusion protein comprising a p66 subunit polypeptide of HIV-1 reverse transcriptase, (ii) a second plasmid which expresses a fusion protein comprising a p51 subunit polypeptide of HIV-1 reverse transcriptase, and (iii) a reporter gene which is activated in the presence of

a complex between the p66 subunit polypeptide and the p51 subunit polypeptide, and determining the level of activity of the reporter gene in the cell in the presence of the compound; and

b) comparing the level of activity of the reporter gene determined in step (a) with a level of activity of the reporter gene determined in the absence of the compound, wherein an increased level of activity of the reporter gene determined in step (a) indicates that the compound is an activator of the formation of the complex between the p51 subunit polypeptide of HIV-1 reverse transcriptase and the p66 subunit polypeptide of HIV-1 reverse transcriptase, thereby indicating that the compound inhibits HIV-1 reverse transcriptase.

4. A method of determining whether a compound enhances formation of a complex between a p66 subunit polypeptide of HIV-1 reverse transcriptase and a p51 subunit polypeptide of HIV-1 reverse transcriptase which comprises:

a) contacting a yeast cell with the compound, which cell comprises (i) a first plasmid which expresses a fusion protein comprising a p66 subunit polypeptide of HIV-1 reverse transcriptase, (ii) a second plasmid which expresses a fusion protein comprising a p51 subunit polypeptide of HIV-1 reverse transcriptase, and (iii) a reporter gene which is activated in the presence of a complex between the p66 subunit polypeptide and the p51 subunit polypeptide, and determining the level of

activity of the reporter gene in the cell in the presence of the compound; and

b) comparing the level of activity of the reporter gene determined in step (a) with a level of activity of the reporter gene determined in the absence of the compound, wherein an increased level of activity of the reporter gene determined in step (a) indicates that the compound is an activator of the formation of the complex between the p51 subunit polypeptide of HIV-1 reverse transcriptase and the p66 subunit polypeptide of HIV-1 reverse transcriptase.

5. A method of determining whether a compound inhibits HIV-1 reverse transcriptase which comprises:

a) contacting a yeast cell with the compound, which cell comprises (i) a first plasmid which expresses a fusion protein comprising a first p66 subunit polypeptide of HIV-1 reverse transcriptase, (ii) a second plasmid which expresses a fusion protein comprising a second p66 subunit polypeptide of HIV-1 reverse transcriptase, and (iii) a reporter gene which is activated in the presence of a complex between the first p66 subunit polypeptide and the second p66 subunit polypeptide, and determining the level of activity of the reporter gene in the cell in the presence of the compound; and

b) comparing the level of activity of the reporter gene determined in step (a) with a level of activity of the reporter gene determined in the absence of the compound, wherein a decreased level of activity of the

reporter gene in step (a) indicates that the compound inhibits formation of a complex between the first p66 subunit polypeptide of HIV-1 reverse transcriptase and the second p66 subunit polypeptide of HIV-1 reverse transcriptase, thereby indicating that the compound inhibits HIV-1 reverse transcriptase.

6. A method of determining whether a compound inhibits formation of a complex between a first p66 subunit polypeptide of HIV-1 reverse transcriptase and a second p66 subunit polypeptide of HIV-1 reverse transcriptase which comprises:

a) contacting a yeast cell with the compound, which cell comprises (i) a first plasmid which expresses a fusion protein comprising a first p66 subunit polypeptide of HIV-1 reverse transcriptase, (ii) a second plasmid which expresses a fusion protein comprising a second p66 subunit polypeptide of HIV-1 reverse transcriptase, and (iii) a reporter gene which is activated in the presence of a complex between the first p66 subunit polypeptide and the second p66 subunit polypeptide, and determining the level of activity of the reporter gene in the cell in the presence of the compound; and

b) comparing the level of activity of the reporter gene determined in step (a) with a level of activity of the reporter gene determined in the absence of the compound, wherein a decreased level of activity of the reporter gene in step (a) indicates that the compound inhibits formation of a complex between the first p66

subunit polypeptide of HIV-1 reverse transcriptase and the second p66 subunit polypeptide of HIV-1 reverse transcriptase.

7. A method of determining whether a compound inhibits HIV-1 reverse transcriptase which comprises:

a) contacting a yeast cell with the compound, which cell comprises (i) a first plasmid which expresses a fusion protein comprising a first p66 subunit polypeptide of HIV-1 reverse transcriptase, (ii) a second plasmid which expresses a fusion protein comprising a second p66 subunit polypeptide of HIV-1 reverse transcriptase, and (iii) a reporter gene which is activated in the presence of a complex between the first p66 subunit polypeptide and the second p66 subunit polypeptide, and determining the level of activity of the reporter gene in the cell in the presence of the compound; and

b) comparing the level of activity of the reporter gene determined in step (a) with a level of activity of the reporter gene determined in the absence of the compound, wherein an increased level of activity of the reporter gene in step (a) indicates that the compound is an activator of the formation of the complex between the first p66 subunit polypeptide of HIV-1 reverse transcriptase and the second p66 subunit polypeptide of HIV-1 reverse transcriptase, thereby indicating that the compound inhibits HIV-1 reverse transcriptase.

8. A method of determining whether a compound enhances formation of a complex between a first p66 subunit polypeptide of HIV-1 reverse transcriptase and a second p66 subunit polypeptide of HIV-1 reverse transcriptase which comprises:

a) contacting a yeast cell with the compound, which cell comprises (i) a first plasmid which expresses a fusion protein comprising a first p66 subunit polypeptide of HIV-1 reverse transcriptase, (ii) a second plasmid which expresses a fusion protein comprising a second p66 subunit polypeptide of HIV-1 reverse transcriptase, and (iii) a reporter gene which is activated in the presence of a complex between the first p66 subunit polypeptide and the second p66 subunit polypeptide, and determining the level of activity of the reporter gene in the cell in the presence of the compound; and

b) comparing the level of activity of the reporter gene determined in step (a) with a level of activity of the reporter gene determined in the absence of the compound, wherein an increased level of activity of the reporter gene in step (a) indicates that the compound is an activator of the formation of the complex between the first p66 subunit polypeptide of HIV-1 reverse transcriptase and the second p66 subunit polypeptide of HIV-1 reverse transcriptase, thereby indicating that the compound inhibits HIV-1 reverse transcriptase.

9. The method of any one of claims 1-8, wherein (a) the

fusion protein expressed by the first plasmid comprises a peptide having a DNA binding domain, and (b) the fusion protein expressed by the second plasmid comprises a peptide having a transcription activation domain.

10. The method of claim 9, wherein the DNA binding domain is a LexA DNA binding domain.

11. The method of claim 10, wherein the peptide having a DNA binding domain comprises LexA amino acid residues 1-87.

12. The method of claim 10, wherein the peptide having a DNA binding domain comprises LexA amino acid residues 1-202.

13. The method of claim 9, wherein the DNA binding domain is a GAL4 DNA binding domain.

14. The method of claim 9, wherein the transcription activation domain is a GAL4 transcription activation domain.

15. The method of claim 14, wherein the peptide having the transcription activation domain comprises GAL4 amino acid residues 768-881.

16. The method of claim 9, wherein the transcription activation domain is a VP16 transcription activation

domain.

17. The method of any one of claims 1-8, wherein (a) the fusion protein expressed by the first plasmid comprises a peptide having a transcription activation domain, and (b) the fusion protein expressed by the second plasmid comprises a peptide having a DNA binding domain.

18. The method of claim 17, wherein the DNA binding domain is a LexA DNA binding domain.

19. The method of claim 18, wherein the peptide having a DNA binding domain comprises LexA amino acid residues 1-87.

20. The method of claim 18, wherein the peptide having a DNA binding domain comprises LexA amino acid residues 1-202.

21. The method of claim 17, wherein the DNA binding domain is a GAL4 DNA binding domain.

22. The method of claim 17, wherein the transcription activation domain is a GAL4 transcription activation domain.

23. The method of claim 22, wherein the transcription activation domain comprises GAL4 amino acid residues 768-881.

24. The method of claim 17, wherein the transcription activation domain is a VP16 transcription activation domain.

25. The method of any one of claims 1-8, wherein the fusion protein expressed by the first plasmid, the second plasmid or both plasmids comprises a peptide comprising consecutive alanine residues.

26. The method of claim 25, wherein the peptide comprising consecutive alanine residues comprises at least 6 alanine residues.

27. The method of any one of claims 1-8, wherein the fusion protein comprises an influenza hemagglutinin (HA) epitope tag.

28. The method of any one of claims 1-8, wherein the reporter gene is a LacZ reporter gene.

29. The method of any one of claims 1-4, wherein (a) the fusion protein expressed by the first plasmid comprises a peptide comprising a LexA protein DNA binding domain, wherein the p66 subunit polypeptide is bound at its C-terminal amino acid to the N-terminal amino acid of the peptide comprising a LexA protein DNA binding domain; and (b) the fusion protein expressed by the second plasmid comprises a Gal4 peptide corresponding to amino acids 768-881 of Gal4, and an influenza hemagglutinin (HA) epitope tag, which

Gal4 peptide is bound at its C-terminal amino acid to the N-terminal amino acid of the influenza hemagglutinin (HA) epitope tag, which influenza hemagglutinin (HA) epitope tag is bound at its C-terminal amino acid to the N-terminal amino acid of the p51 subunit polypeptide.

30. The method of any one of claims 1-4, wherein (a) the fusion protein expressed by the first plasmid comprises a peptide comprising a LexA protein DNA binding domain, wherein the p66 subunit polypeptide is bound at it's C-terminal amino acid to the N-terminal amino acid of the peptide comprising a LexA protein DNA binding domain; and (b) the fusion protein expressed by the second plasmid comprises a Gal4 peptide corresponding to amino acids 768-881 of Gal4, which Gal4 peptide is bound at its C-terminal amino acid to the N-terminal amino acid of the p51 subunit polypeptide.

31. The method of any one of claims 1-4, wherein (a) the fusion protein expressed by the first plasmid comprises a LexA peptide corresponding to amino acid residues 1-87, wherein the LexA peptide is bound at its C-terminal amino acid to the N-terminal amino acid of the of the p66 subunit polypeptide; and (b) the fusion protein expressed by the second plasmid comprises a Gal4 peptide corresponding to amino acids 768-881 of Gal4, and an influenza hemagglutinin (HA) epitope tag, which Gal4 peptide is bound at its C-

terminal amino acid to the N-terminal amino acid of the influenza hemagglutinin (HA) epitope tag, which influenza hemagglutinin (HA) epitope tag is bound at its C-terminal amino acid to the N-terminal amino acid of the p51 subunit polypeptide.

32. The method of any one of claims 1-4, wherein (a) the fusion protein expressed by the first plasmid comprises a LexA peptide corresponding to amino acid residues 1-87, wherein the LexA peptide is bound at its C-terminal amino acid to the N-terminal amino acid of the of the p66 subunit polypeptide; and (b) the fusion protein expressed by the second plasmid comprises a Gal4 peptide corresponding to amino acids 768-881 of Gal4, which Gal4 peptide is bound at its C-terminal amino acid to the N-terminal amino acid of the p51 subunit polypeptide.

33. The method of any one of claims 1-4, wherein (a) the fusion protein expressed by the first plasmid comprises a LexA peptide corresponding to amino acid residues 1-202, and a peptide comprising six consecutive alanine residues, wherein the LexA peptide is bound at its C-terminal amino acid to the N-terminal amino acid of the peptide comprising six consecutive alanine residues, wherein the peptide comprising six consecutive alanine residues is bound at its C-terminal amino acid to the N-terminal amino acid of the p66 subunit polypeptide; and (b) the fusion protein expressed by the second plasmid

comprises a Gal4 peptide corresponding to amino acids 768-881 of Gal4, which Gal4 peptide is bound at its C-terminal amino acid to the N-terminal amino acid of the p51 subunit polypeptide.

34. The method of any one of claims 1-4, wherein (a) the fusion protein expressed by the first plasmid comprises a LexA peptide corresponding to amino acid residues 1-202, and a peptide comprising six consecutive alanine residues, wherein the LexA peptide is bound at its C-terminal amino acid to the N-terminal amino acid of the peptide comprising six consecutive alanine residues, wherein the peptide comprising six consecutive alanine residues is bound at its C-terminal amino acid to the N-terminal amino acid of the p66 subunit polypeptide; and (b) the fusion protein expressed by the second plasmid comprises a Gal4 peptide corresponding to amino acids 768-881 of Gal4, and an influenza hemagglutinin (HA) epitope tag, which Gal4 peptide is bound at its C-terminal amino acid to the N-terminal amino acid of the influenza hemagglutinin (HA) epitope tag, which influenza hemagglutinin (HA) epitope tag is bound at its C-terminal amino acid to the N-terminal amino acid of the p51 subunit polypeptide.

35. The method of any one of claims 1-4, wherein (a) the fusion protein expressed by the first plasmid comprises a Gal4 peptide corresponding to amino acids 768-881 of Gal4, an influenza hemagglutinin (HA)

epitope tag, and a peptide comprising six consecutive alanine residues, wherein the Gal4 peptide is bound at its C-terminal amino acid to the N-terminal amino acid of the influenza hemagglutinin (HA) epitope tag, wherein the influenza hemagglutinin (HA) epitope tag is bound at its C-terminal amino acid to the N-terminal amino acid of the peptide comprising six consecutive alanine residues, wherein the peptide comprising six consecutive alanine residues is bound at its C-terminal amino acid to the N-terminal amino acid of the p66 subunit polypeptide; and (b) the fusion protein expressed by second plasmid comprises a peptide comprising a LexA protein DNA binding domain, wherein the p51 subunit polypeptide is bound at its C-terminal amino acid to the N-terminal amino acid of the peptide comprising a LexA protein DNA binding domain.

36. The method of any one of claims 1-4, wherein (a) the fusion protein expressed by the first plasmid comprises a Gal4 peptide corresponding to amino acids 768-881 of Gal4, an influenza hemagglutinin (HA) epitope tag, and a peptide comprising six consecutive alanine residues, wherein the Gal4 peptide is bound at its C-terminal amino acid to the N-terminal amino acid of the influenza hemagglutinin (HA) epitope tag, wherein the influenza hemagglutinin (HA) epitope tag is bound at its C-terminal amino acid to the N-terminal amino acid of the peptide comprising six consecutive alanine residues, wherein the peptide

comprising six consecutive alanine residues is bound at its C-terminal amino acid to the N-terminal amino acid of the p66 subunit polypeptide; and (b) the fusion protein expressed by second plasmid comprises a peptide comprising a LexA protein DNA binding domain, wherein peptide comprising a LexA protein DNA binding domain is bound at its C-terminal amino acid to the N-terminal amino acid of the p51 subunit polypeptide.

37. The method of any one of claims 1-4, wherein (a) the fusion protein expressed by the first plasmid comprises a Gal4 peptide corresponding to amino acids 768-881 of Gal4, an influenza hemagglutinin (HA) epitope tag, and a peptide comprising six consecutive alanine residues, wherein the Gal4 peptide is bound at its C-terminal amino acid to the N-terminal amino acid of the influenza hemagglutinin (HA) epitope tag, wherein the influenza hemagglutinin (HA) epitope tag is bound at its C-terminal amino acid to the N-terminal amino acid of the peptide comprising six consecutive alanine residues, wherein the peptide comprising six consecutive alanine residues is bound at its C-terminal amino acid to the N-terminal amino acid of the p66 subunit polypeptide; and (b) the fusion protein expressed by second plasmid comprises a peptide comprising a Gal4 protein DNA binding domain, which peptide comprising a Gal4 protein DNA binding domain is bound at its C-terminal amino acid to the N-terminal amino acid of the p51 subunit

polypeptide.

38. The method of any one of claims 1-4, wherein (a) the fusion protein expressed by the first plasmid comprises a Gal4 peptide corresponding to amino acids 768-881 of Gal4, wherein the Gal4 peptide is bound at its C-terminal amino acid to the N-terminal amino acid of the p66 subunit polypeptide; and (b) the fusion protein expressed by second plasmid comprises a peptide comprising a LexA protein DNA binding domain, wherein the p51 subunit polypeptide is bound at its C-terminal amino acid to the N-terminal amino acid of the peptide comprising a LexA protein DNA binding domain.

39. The method of any one of claims 1-4, wherein (a) the fusion protein expressed by the first plasmid comprises a Gal4 peptide corresponding to amino acids 768-881 of Gal4, wherein the Gal4 peptide is bound at its C-terminal amino acid to the N-terminal amino acid of the p66 subunit polypeptide; and (b) the fusion protein expressed by second plasmid comprises a peptide comprising a LexA protein DNA binding domain, which peptide comprising a LexA protein DNA binding domain is bound at its C-terminal amino acid to the N-terminal amino acid of the p51 subunit polypeptide.

40. The method of any one of claims 1-4, wherein (a) the fusion protein expressed by the first plasmid comprises a Gal4 peptide corresponding to amino acids

768-881 of Gal4, wherein the Gal4 peptide is bound at its C-terminal amino acid to the N-terminal amino acid of the p66 subunit polypeptide; and (b) the fusion protein expressed by second plasmid comprises a peptide comprising a Gal4 protein DNA binding domain, which peptide comprising a Gal4 protein DNA binding domain is bound at its C-terminal amino acid to the N-terminal amino acid of the p51 subunit polypeptide.

41. The method of any one of claims 5-8, wherein (a) the fusion protein expressed by the first plasmid comprises a peptide comprising a LexA protein DNA binding domain, wherein the p66 subunit polypeptide is bound at it's C-terminal amino acid to the N-terminal amino acid of the peptide comprising a LexA protein DNA binding domain; and (b) the fusion protein expressed by the second plasmid comprises a Gal4 peptide corresponding to amino acids 768-881 of Gal4, an influenza hemagglutinin (HA) epitope tag, and a peptide comprising six consecutive alanine residues, wherein the Gal4 peptide is bound at its C-terminal amino acid to the N-terminal amino acid of the influenza hemagglutinin (HA) epitope tag, wherein the influenza hemagglutinin (HA) epitope tag is bound at its C-terminal amino acid to the N-terminal amino acid of the peptide comprising six consecutive alanine residues, wherein the peptide comprising six consecutive alanine residues is bound at its C-terminal amino acid to the N-terminal amino acid of the p66 subunit polypeptide.

42. A method of making a pharmaceutical composition which comprises:

a) determining whether a compound inhibits HIV-1 reverse transcriptase by the method of any one of claims 1-8;

b) recovering the compound if it is determined to inhibit HIV-1 reverse transcriptase; and

c) admixing the compound with a pharmaceutically acceptable carrier.

43. A method of inhibiting formation of a complex between the p51 subunit polypeptide of HIV-1 reverse transcriptase and a p66 subunit polypeptide of HIV-1 reverse transcriptase, which comprises contacting either (1) the p51 subunit polypeptide, (2) the p66 subunit polypeptide, or (3) both the p51 subunit polypeptide and the p66 subunit polypeptide, with an effective amount of a compound determined to do so by the method of claim 2, so to thereby inhibit formation of a complex between the p51 subunit polypeptide of HIV-1 reverse transcriptase and a p66 subunit polypeptide of HIV-1 reverse transcriptase.

44. A method of enhancing formation of a complex between the p51 subunit polypeptide of HIV-1 reverse transcriptase and a p66 subunit polypeptide of HIV-1 reverse transcriptase, which comprises contacting either (1) the p51 subunit polypeptide, (2) the p66 subunit polypeptide, or (3) both the p51 subunit polypeptide and the p66 subunit polypeptide, with an

effective amount of a compound determined to do so by the method of claim 4, so to thereby enhance formation of a complex between the p51 subunit polypeptide of HIV-1 reverse transcriptase and a p66 subunit polypeptide of HIV-1 reverse transcriptase.

45. A method of inhibiting formation of a complex between a first p66 subunit polypeptide of HIV-1 reverse transcriptase and a second p66 subunit polypeptide of HIV-1 reverse transcriptase, which comprises contacting either (1) the first p66 subunit polypeptide, (2) the second p66 subunit polypeptide, or (3) both the first p66 subunit polypeptide and the second p66 subunit polypeptide, with an effective amount of a compound determined to do so by the method of claim 6, so to thereby inhibit formation of a complex between the first p66 subunit polypeptide of HIV-1 reverse transcriptase and the second p66 subunit polypeptide of HIV-1 reverse transcriptase.

46. A method of enhancing formation of a complex between a first p66 subunit polypeptide of HIV-1 reverse transcriptase and a second p66 subunit polypeptide of HIV-1 reverse transcriptase, which comprises contacting either (1) the first p66 subunit polypeptide, (2) the second p66 subunit polypeptide, or (3) both the first p66 subunit polypeptide and the second p66 subunit polypeptide, with an effective amount of a compound determined to do so by the method of claim 8, so to thereby enhance formation of a

complex between the first p66 subunit polypeptide of HIV-1 reverse transcriptase and the second p66 subunit polypeptide of HIV-1 reverse transcriptase.

47. The method of any one of claims 43-46, wherein the HIV-1 reverse transcriptase is present in a subject and the contacting is effected by administering the compound to the subject.

48. The method of claim 47, wherein the compound is administered orally, intravenously, subcutaneously, intramuscularly, topically or by liposome-mediated delivery.

49. The method of claim 47, wherein the subject is a human being, a primate, an equine, an opine, an avian, a bovine, a porcine, a canine, a feline or a mouse.

50. The method of claim 47, wherein the effective amount of the compound is between about 1mg and about 50mg per kg body weight of the subject.

51. The method of claim 50, wherein the effective amount of the compound is between about 2mg and about 40mg per kg body weight of the subject.

52. The method of claim 51, wherein the effective amount of the compound is between about 3mg and about 30mg per kg body weight of the subject.

53. The method of claim 52, wherein the effective amount of the compound is between about 4mg and about 20mg per kg body weight of the subject.

54. The method of claim 53, wherein the effective amount of the compound is between about 5mg and about 10mg per kg body weight of the subject.

55. The method of claim 54, wherein the compound is administered at least once per day.

56. The method of claim 47, wherein the compound is administered daily.

57. The method of claim 47, wherein the compound is administered every other day.

58. The method of claim 47, wherein the compound is administered every 6 to 8 days.

59. The method of claim 47, wherein the compound is administered weekly.

60. A compound determined to be capable of inhibiting formation of a complex between a p51 subunit polypeptide of HIV-1 reverse transcriptase and a p66 subunit polypeptide of HIV-1 reverse transcriptase by the method of claim 2.

61. A compound determined to be capable of enhancing

formation of a complex between a p51 subunit polypeptide of HIV-1 reverse transcriptase and a p66 subunit polypeptide of HIV-1 reverse transcriptase by the method of claim 4.

62. A compound determined to be capable of inhibiting formation of a complex between a first p66 subunit polypeptide of HIV-1 reverse transcriptase and a second p66 subunit polypeptide of HIV-1 reverse transcriptase by the method of claim 6.

63. A compound determined to be capable of enhancing formation of a complex between a first p66 subunit polypeptide of HIV-1 reverse transcriptase and a second p66 subunit polypeptide of HIV-1 reverse transcriptase by the method of claim 8.

64. A composition which comprises the compound of any one of claims 60-63 and a carrier.